

Case Report

INFLAMMATORY LENS DEPOSITION FOLLOWING SUPRAMID RIPCORD REMOVAL

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ARTICLE INFO

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Received:
November 2021
Accepted for publication:
December 2021

Keywords:

glaucoma drainage device;
intraluminal supramid stent;
ripcord suture removal;
ocular hypotony intraocular inflammation;
uveitis

ABSTRACT

This is a case report of anterior chamber inflammation after removal of Supramid[®] intraluminal Stent suture used to prevent postoperative of hypotony in a patient with non-valved Glaucoma drainage device (GDD). Removal of Ripcord Supramid[®] intraluminal stent from glaucoma drainage device caused sudden ocular decompression and hypotony; this induced breakdown in the blood-aqueous barrier (BAB) causing anterior uveitis and deposition on the anterior surface of the intraocular lens. In conclusion, hypotony following removal of the intraluminal Supramid[®] stent result in intraocular inflammation.

INTRODUCTION

Intraocular damage caused by uveitis can result in increased intraocular pressure (IOP), with subsequent damage to the optic nerve [1]. Several factors may be involved in the pathogenesis of inflammatory glaucoma, including obstruction of the trabecular meshwork by inflammatory cells and proteins, trabeculitis (inflammation of the trabecular meshwork), formation of peripheral anterior synechiae, posterior synechia leading to pupillary block, iris neovascularization and neovascular glaucoma, and anterior rotation of the lens-iris diaphragm [2]. Additionally, the use of steroids to control the inflammatory process may cause secondary elevation of IOP. Inflammation associated IOP elevation and glaucoma may become a severe complication and contribute to visual loss in patients with uveitis [3].

Glaucoma is defined as the presence of either pathologic cupping of the optic disc and/or a glaucomatous visual field defect with elevated IOP above 21 mm Hg [4]. The only known modifiable factor is intraocular pressure (IOP). Therefore treatment is focused on lowering IOP through medical or surgical therapy. Surgical therapy is indicated when medical or laser therapies fail to provide adequate IOP control to slow down the progressive optic nerve damage [5].

Tube shunt surgery has a higher success rate compared to trabeculectomy during the follow-up period [5]. It is also indicated in patients with refractory glaucoma which have a high risk of trabeculectomy failure [5]. Glaucoma drainage devices (GDDs) are composed of a silicone tube (~0.3 mm inner diameter) connected to a plate that drains aqueous humor from the anterior chamber to the subconjunctival space. Glaucoma drainage devices can be valved to restrict flow below a certain level of IOP, such as the Ahmed glaucoma valve (New World Medical, Rancho Cucamonga, CA), or non-valved such as the 350 mm² Baerveldt glaucoma implant (BGI Abbott Medical Optics, Santa Ana, CA).

Studies have shown that the BGI has comparable outcomes to glaucoma filtration surgery [6]. The BGI requires the use of a stent suture inserted within or around the tube lumen to restrict the aqueous flow initially following device insertion in order to prevent the initial hypotony. The stent is often removed several months later [6]. We report a case of intraocular inflammation after supramid ripcord removal causing inflammatory lens deposition in a young man with advanced steroid-induced glaucoma requiring BGI implantation.

CASE REPORT

A 44-year-old Malay gentleman, with a history of bilateral idiopathic anterior uveitis was referred to our glaucoma clinic for further management. The patient has glaucoma secondary to anterior uveitis, and is also known to be a steroid responder causing bilateral advanced glaucoma. He has been on treatment for glaucoma in the last 20 years.

He underwent trabeculectomy surgery on the right eye twice and left eye once. Intraocular pressure in the left eye was controlled after the trabeculectomy throughout, while the right eye require multiple 5FU needling and antiglaucoma medications. Despite that IOP in the right eye was uncontrolled with IOP as high as 56mmHg.

A Xen tube insertion was initially performed 3 years prior. Over a period of 2 years after Xen tube insertion, IOP in the right eye was well controlled until subconjunctival fibrosis limits the IOP control, despite multiple 5FU needling and antiglaucoma medication. Hence, the decision for Baerveldt

implantation was made. The intraluminal stent was inserted into the drainage tube using Supramid^R 3-0 suture. The stent was removed three months later to allow better IOP control.

Five months post stent removal patient presented with a complaint of worsening right visual acuity, with best-corrected visual acuity (BCVA) of 1/60, a drop from 6/24. Examination showed multiple deposits and fibrin on the anterior surface of his intraocular lens, with a significant presence of giant cells (Figure 1). However, no anterior chamber cells or flare were observed. The IOP was 10mm Hg. Topical corticosteroid, dexamethasone 0.1 % four times a day was started to treat the inflammation.

On a subsequent visit to the eye clinic 2 weeks later, the patient claimed that his right vision was better. His right BCVA improved to 6/60. The deposits on the anterior surface of the intraocular lens were notably lesser (Figure 2a). There was no anterior chamber cells or flare. His right BCVA further improved to 6/28, slightly less than his best visual acuity 2 weeks later (Figure 2b).

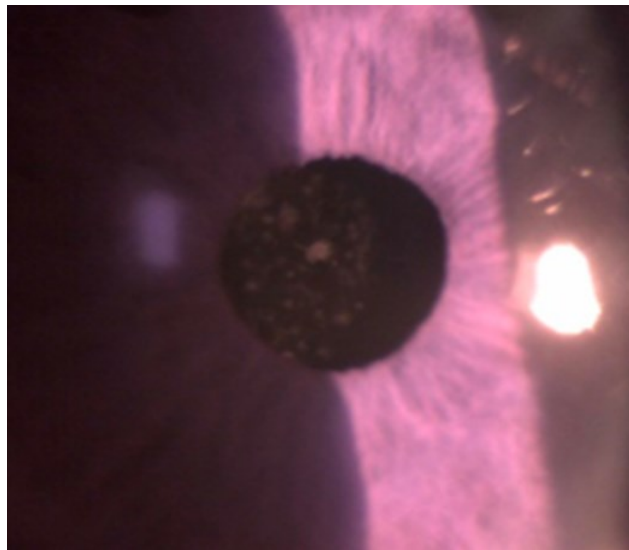


Figure 1: Presence of deposit and fibrin on the anterior intra ocular lens.

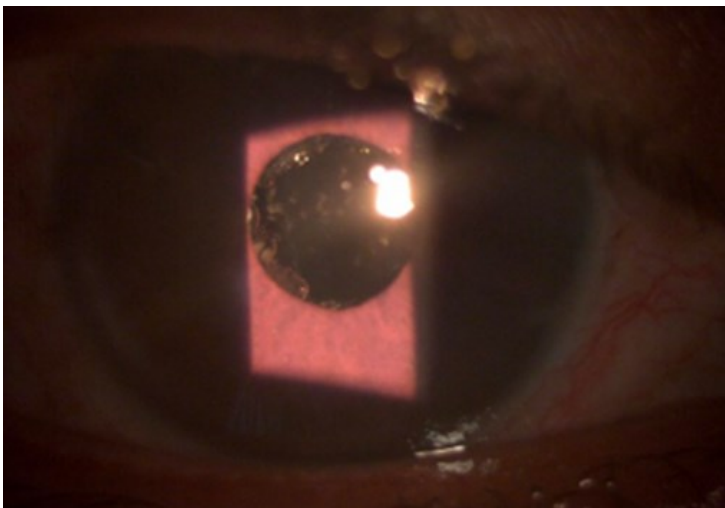


Figure 2a: Improved intraocular inflammation with less deposits on the anterior surface of the intraocular lens.



Figure 2b: Notable lesser deposits on the anterior surface of the intraocular lens.

DISCUSSION

This case report highlights the occurrence of intraocular inflammation and intraocular lens deposit induced by removal of supramid stent from the Baerveldt tube using a ripcord technique. We think this is probably attributed by the disturbance in blood ocular barrier induced by the removal of the supramid stent which caused relative hypotony. The blood-ocular barrier consists of blood aqueous barrier (BAB) and blood-retinal barrier (BRB). The BAB is composed of the endothelial cells of the blood vessels in the iris and the non-pigmented cell layer of the ciliary epithelium [7]. The Blood retinal barrier (BRB) is formed by tight junctions between the endothelial cells of the retinal vessels (the inner BRB) and by similar tight junctions in the retinal pigment epithelium (the outer BRB) [7].

Ocular hypotony is known to cause a breakdown of the blood-aqueous barrier by the opening of the non-fenestrated endothelial layer of the iris vessels and the intercellular junctions of the ciliary body, alongside an increase in episcleral venous pressure. In addition, ocular hypotony leads to alterations in the intercellular tight junctions of the RPE and vascular endothelial cells, resulting in a breakdown of both the inner and outer blood-retinal barriers [8].

The breakdown of blood-ocular barriers allows inflammatory mediators, cells and proteins to enter into intraocular chambers and ocular tissues. Eyes with anterior uveitis have a reduction of the intercellular junctions in the ciliary epithelium. In addition, junctional disruptions, likely resulting in an increase in vascular permeability have been found in inter-endothelial junctions of the iris vessels in the eyes [8]. Macrophages, polymorphonuclear leukocytes and lymphocytes can thus elicit the inflammatory response and play a key role in the initiation of the uveitis process [8].

Deposits form on the intraocular lens in our case, were likely giant cells, visible on slit lamps as sharp, round, or oval spots with or without pigmentation [9]. Of note, a good response was seen in this patient towards topical steroids in resolving the intraocular lens deposit, as topical corticosteroids have been shown to facilitate and improve the blood aqueous barrier [10]. The use of topical corticosteroids as prophylaxis prior intraluminal stent removal is highly recommended to reduce the possible inflammation following the procedure which could worsen the visual outcome.

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